INFLUENZA C IN A NAVAL RECRUIT POPULATION

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IN the spring of 1958, an outbreak of adenovirus infection occurred in the Portsmouth Naval Command (Fraser and Hatch 1959). Recruits at a training establishment in the area were also affected. This centre (V) takes into the Navy each month a group of boys aged 15–16. After one month in this establishment a small number are transferred to another training centre (G) in Suffolk. Further studies at these recruit centres showed that a considerable number of new recruits had acquired fourfold or greater rises in complement-fixing antibody titres against influenza-virus C.

Clinical and Laboratory Methods

At centre V a card was made out for each recruit who joined, and on it was recorded each illness which caused the recruit to seek medical advice. Since the recruits were young, it was thought that most of those who had upper-respiratory infections would report their illness. It was considered that even minor illnesses such as "colds" would be reported.

A small sample of blood was obtained from each recruit on the day he joined and three months later, so that his blood-group could be determined and confirmed. Sera from these blood samples were stored at −15°C until they were tested in pairs by complement-fixation against antigens of influenza viruses A, B, and C, adenovirus, psittacosis/lymphogranuloma-venereum (L.G.V.) group of viruses, and *Rickettsia burneti* (Fraser and Hatch 1959). Fourfold or greater rises in titre were considered significant. All other titres were considered negative.

Clinical and Laboratory Findings

452 boys joined the training centre V between May and October, 1958. 60 moved to the training centre G after one month's initial training.

379 pairs of sera were collected and tested from the 392 boys who stayed at V for the whole three-month period, and 65 (17%) showed significant rises in titre to influenza-virus C. The distribution of these is shown in table I. 6 of those who had rises against influenza-virus C also had significant rises against other antigens; 2 were against adenovirus, 1 against influenza-virus A, 2 against the psittacosis-L.G.V. group of viruses, and 1 against influenza-virus A and the psittacosis-L.G.V. group. 269 pairs were negative, and 45 had significant rises against antigens other than influenza-virus C.

Only 3 (5%) of the 60 boys who transferred to G showed significant rises against influenza-virus C. These are shown in table II; 6 had significant rises against other antigens, and 51 were negative.

Taking both groups together, there were 62 boys who had rises against influenza-virus C only. 26 (42%) of these reported sick with an upper-respiratory infection during the period of study; of these 26, 17 had sore throat and mild dysphagia and 10 had fever. Of the 6 with multiple infections, 2 had a history of upper-respiratory infection and 1 was febrile.

320 recruits in all were serologically negative in the same period. 94 (29%) of these had upper-respiratory infections, and 19 of these had fever.

During the period May to December, 1958, a further 289 pairs of sera were obtained from patients admitted to hospital with upper-respiratory illnesses from other units in the Command, and all were negative for influenza-virus C.

Discussion

In this country, Andrews and McDonald (1955) showed that complement-fixing antibodies against influenza-virus C were widely distributed throughout the population;

TABLE I—DISTRIBUTION OF FOURFOLD OR GREATER RISES IN TITRE AGAINST INFLUENZA-VIRUS C FOUND IN 65 OF 379 PAIRS OF SERA OBTAINED FROM BOYS AT TRAINING CENTRE V

Entry no.	No. tested	No. in class	Dates sera taken (1958)	Serum titres								Total	% of those tested
				<1/5–1/10	<1/5–1/20	<1/5–1/40	1/5–1/20	1/5–1/40	<1/10–1/20	<1/10–1/40	<1/10–1/80		
13	79	84	May 6, Aug. 7	6	1	..	8	15	..	..	..	30	38
14	57	60	June 10, Sept. 15	4	5	2	2	1	1	0	1	16	28
15	68	69	July 15, Oct. 14	..	..	..	..	..	5	1	8	14	20
16	102	105	Sept. 2, Dec. 2	..	1	..	..	..	..	2	..	3	3
17	73	74	Oct. 7, Jan. 12, 1959	1	..	..	..	..	1	..	..	2	3
Totals	379	392		11	7	2	10	16	7	3	9	65	17

TABLE II—DISTRIBUTION OF FOURFOLD OR GREATER RISES IN TITRE AGAINST INFLUENZA-VIRUS C FOUND IN 3 OF 60 PAIRS OF SERA OBTAINED FROM BOYS WHO JOINED CENTRE V BUT TRANSFERRED TO CENTRE G AFTER ONE MONTH

Entry no.	No. tested	No. in class	Dates sera taken (1958)	Serum titres								Total	% of those tested
				<1/5–1/10	<1/5–1/20	<1/5–1/40	1/5–1/20	1/5–1/40	<1/10–1/20	<1/10–1/40	<1/10–1/80		
13	20	20	May 6, Aug.	..	..	..	..	..	..	..	..	0	0
14	17	17	June 10, Sept.	1	..	..	..	..	1	1	..	3	18
15	2	2	July 15, Oct.	..	..	..	..	..	..	..	..	0	0
16	13	13	Sept. 2, Dec.	..	..	..	..	..	..	..	..	0	0
17	8	8	Oct. 7, Jan., 1959	..	..	..	..	..	..	..	..	0	0
Totals	60	60		1	0	0	0	0	1	1	0	3	5

they concluded that infection by this virus was common and that it might be responsible for acute respiratory illness. Grist (1955) reported that infection often occurred in childhood and early adult life.

This study has shown that 68 (15%) of 439 boy recruits who were tested had acquired significant rises in antibody against influenza C and had been infected between May, 1958, and January, 1959. Table I shows that most of the infections occurred in those who joined in May, June, and July, and that between 1 in 3 and 1 in 5 were affected in these three groups. It is noticeable that the number of cases declined immediately after August and that the epidemic was virtually over by the end of July. This may have been due to dispersal during the leave period or to the fact that no new recruits were taken into the centre during August.

The numbers of those who transferred to G are too small for direct comparison. Nevertheless, the fact that only 3 cases of influenza C were found, all in the group which joined V in June and transferred to G in July, suggests that most of the infections at V occurred in June.

The illness experienced by this recruit population appeared to be mild since less than half those who had influenza C were ill enough to report sick. This may explain why there is so little information available about this disease.

On the other hand, more serious illnesses, believed to be due to influenza-virus C, have been reported by Gerber et al. (1952) and Librach (1956), and influenza-virus C was isolated from a fatal case of pneumonia in 1956 by Darke et al. (1957).

There is little information in this study which indicated the degree of severity of the illness in any particular individual. It is tempting to dismiss the infection as trivial, but it would be unwise to draw this conclusion without further evidence than is presented here.

Summary

Sera were obtained from 439 boys on the day they joined a training establishment and again three months later.

68 showed significant rises in complement-fixing antibody titres to influenza virus C, and 6 of these also had rises against other viruses.

Less than half those who had influenza C were sufficiently ill to seek medical advice.

We are indebted to the Medical Director-General of the Navy for permission to publish, and to Prof. A. Bradford Hill for statistical advice. Surgeon Captain J. C. Gent, Q.H.P., H.M.S. *Ganges*, kindly allowed us access to the records of the boys transferred to centre G and supplied samples of blood.

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Addendum

Paired sera were tested from 82 of 83 and 87 of 89 boys who joined centre V in November, 1958, and January 1959, respectively. 1 boy who joined in November and transferred to centre G one month later had a significant rise in titre to both influenza-virus C and the psittacosis-L.G.V. group of viruses; all the others were negative for influenza-virus C.

A DEPOT LEPROMIN TEST AND B.C.G. VACCINATION

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Kensuke Mitsuda reported, first in 1916 and again in 1932, that intracutaneous injection of a boiled emulsion of bacteria obtained from leprous nodules usually gave a negative reaction in nodular leprosy (lepromatous) but a positive reaction in neuromacular leprosy (tuberculoid) (Hayashi 1933). Failure to obtain the bacillus in pure culture has hindered preparation of a more refined antigen, and the lepromin in common use is made by grinding 1 g. of autoclaved bacteriologically positive tissue in 20 ml. of isotonic saline. Intradermal injection of 0·1 ml. is followed in most non-lepromatous patients by a tuberculin-like response in twenty-four to forty-eight hours. This rapidly subsides to be succeeded by a nodular infiltration which reaches a maximum in three to four weeks. The late Mitsuda reaction is believed to depend on the presence of whole bacilli, is more reliable than the early response, and is used in classification and prognosis. The late reaction is the one referred to in this paper. It is graded as follows:

Infiltrations of 1 and 2 mm.	Doubtful
Infiltrations of 3 and 4 mm.	Weakly positive
Infiltrations of 5 mm. or more	Positive (fig. 1)
Infiltration with ulceration	Strongly positive (fig. 2)

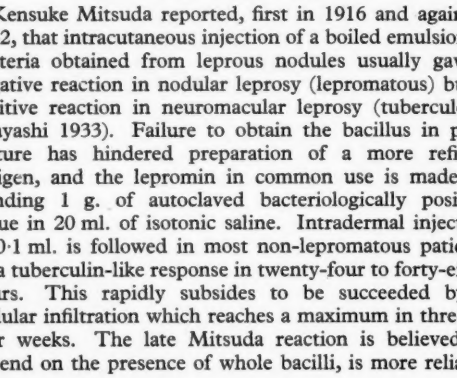

Fig. 1.

For the purposes of preventive medicine, the population of an endemic area can be divided into weak or negative

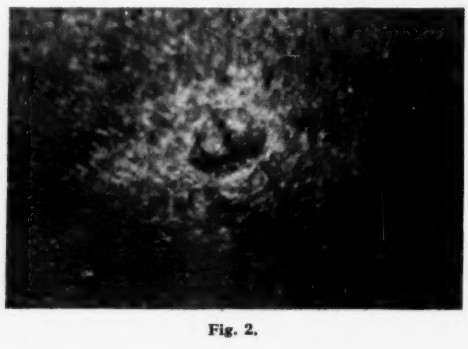

Fig. 2.

reactors with an infiltration less than 5 mm., and strong reactors with an infiltration of 5 mm. or more. The weak reactors will not necessarily develop leprosy if exposed to infection; nor will the strongly positive always escape; but, in general, those with a weak response develop the more serious and infectious lesions whilst the strong reactors develop the milder self-healing ones. The interesting discovery that B.C.G. vaccination is followed by conversion in the majority of lepromin-negative people has made it even more important to be able to identify the weak reactors among the children and contacts of patients.

The lepromin test cannot be widely used, because the supply of antigen is insufficient. This is restricted by the number of suitable lepromatous patients, which in some areas is being progressively reduced by modern treatment. But even if enough antigen could be obtained, it is doubtful whether the general public would submit to the test in its present form, because the strongest reactors develop indolent ulcers at the site of injection (fig. 2) and, ironically enough, they are the ones least at risk, whom it is therefore least important to discover and protect.

Modification of Test

Various attempts have been made to substitute a more dilute antigen and to devise a simple method of stating the results in terms of those that would have been expected with normal antigen. The work of Floch (1954, 1955), Diniz and Neto (1954), and Schujman (1955) was particularly important, and reference was made to the necessity of a more economical test by Wade (1954). More recently it was suggested that the same grading of positives might be achieved with 1/100 lepromin if the dividing line between weak and strong reactors was reduced to 4 mm. (infiltrations of 1 and 2 mm., doubtful; 3 mm., weakly positive; 4 mm. or more, positive; ulceration, strongly positive). Ulceration was still an undesirable complication, however, and an antigen dilute enough to avoid it was not sensitive enough in the lower grades. Accordingly we have tried a modification of the test, using the Heaf multi-puncture apparatus instead of an intradermal injection (Kinnear Brown 1958).

With this method there was no distinctive early and late reaction, although in a few patients one appeared in the first week and faded after a few days. In lepromin-positive patients the normal sequel was the formation of a ring of six small papules, one at the site of each puncture. These persisted and reached their maximum about the end of the third week.

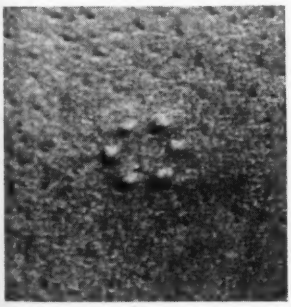

Fig. 3.

The reactions were definite and unmistakable but smaller than with tuberculin. After a series of trials it was found possible to grade them according to the size of the papules. In making comparisons, the extent to which the interval between adjacent papules was diminished was an additional help. A ring of hyperpigmented macules of pinhead size was disregarded. The scale finally adopted was:

Grade I.—Four or more palpable and discrete papules.

Grade II.—Four or more prominent and discrete papules (fig. 3).

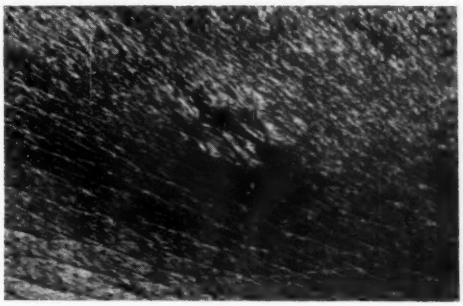

Fig. 4.

Grade III.—Four or more prominent papules with pinpoint vesiculation, or four or more papules tending to coalesce (fig. 4).

A reaction with fewer than six papules was usually the result of uneven pressure of the endplate of the apparatus on the skin.

Comparisons

Patients known to be positive were then tested by the intradermal and multipuncture routes with 1/20 lepromin.

When the two tests were applied at widely separated points—e.g., the forearm and the back—or at an adequate time-interval, the results correlated so well that it was felt for some purposes, and especially in the field, the multipuncture test could with advantage be substituted for the classical Mitsuda method. (It is worth noting, however, that in some patients, when the tests were made simultaneously and near together, the intradermal response—i.e., to the larger dose—appeared to suppress the response to the antigen administered by multipuncture. The same phenomenon occurred only very occasionally when the tests were made simultaneously but at a distance.) With 1/100 lepromin there was a fall in the number of positives. When a 1/20 depot lepromin was substituted, prepared similarly to the depot tuberculin described by James and Pepys (1956), and given by the multipuncture route, the response corresponded completely with the response to ordinary lepromin however administered. (The depot medium consisted of 1 part anhydrous lanolin and 8 parts of light liquid paraffin to which was added 20% of isotonic saline solution.)

Comparison of a series of positives, in which the results to 1/20 and 1/100 normal lepromin had been determined, with a similar series in which the same strengths of depot lepromin had been used, showed a much smaller reduction of positives with the 1/100 depot preparation. (The series were small but the percentage reductions were 47 and 25.) The reactions to the 1/100 depot lepromin were, if anything, more emphatic, and the proportion that responded to a 1/500 depot lepromin in the latter series was at least as great as that which reacted to 1/100 normal lepromin in the former. It should, perhaps, be added that it is difficult to use the same patients all the time, because simultaneous tests can interfere with each other (with the multipuncture route this is much less noticeable) and repeated lepromin tests may increase sensitivity.

Three years ago we had noticed that the addition of 10% of glycerin, as in purified protein derivative used in the Heaf tuberculin test, often enhanced the response to multipuncture lepromin (Kinnear Brown 1958). Floch (1955) had reported that a 1/750 lepromin to which 12% glycerin and 2% paraffin had been added gave a stronger reaction in half his patients. Repeated trials have now convinced us that by the multipuncture route a depot lepromin is, for most purposes, as efficient as a saline lepromin in the same concentration; also it is easier to prepare, more economical, and never inconvenient to the patient. A 1/100 depot lepromin gives slightly more

negatives than 1/20, but this would be an advantage in field work because the increase consists of weak reactors who ought to be given the benefit of any protection that is possible. Using a tuberculin syringe with platinum iridium needle (size S.W.G. 17) as dropper, so that the smallest drop but not the needle touched the sterilised skin, we found it was possible to get 25 multipuncture tests from 0·1 ml. of 1/100 depot lepromin. 1 g. of autoclaved tissue should, therefore, provide enough depot lepromin in this strength for more than 20,000 tests, compared with 200 by the usual intradermal injection—a tremendous economy where discovery of the weak reactor is the main objective.

Depot Lepromin and B.C.G.

It remained to be seen whether, having identified the weak and negative responders, a depot lepromin would persist in the skin sufficiently long to indicate the effect of subsequent B.C.G. vaccination.

10 children about a year old, in contact with their mothers who were suffering from leprosy, were tested with depot lepromin in concentrations of 1/20 and 1/100. 1 was positive to both; after five weeks the others were still negative. They were then tested with tuberculin and all shown to be negative. As the tuberculin tests were read, they were vaccinated with B.C.G., and retested with 1/20 depot lepromin, which eventually indicated lepromin conversion in the nine who had been negative. 1 child showed signs of conversion three weeks after vaccination, in the others they appeared a week later. The original lepromin tests did not show any change for another two weeks when 7 became positive in both concentrations. In the other 2 there was an alteration at the site of the tests but the change was less definite.

In at least 7, the original depot lepromin remained in the skin long enough to act as an indicator. This might have happened in all if the interval between the first tests and the vaccination had been less. The long interval was chosen deliberately. The fact that some of the depot remained in situ all the time although probably in a diminishing amount, and that the early tests were the last to change, suggests that the conversion was due not to sensitisation by the original dose of lepromin but to the vaccination.

Subsequently a number of healthy schoolchildren, negative to lepromin and tuberculin, were vaccinated after a three-weeks interval, with the following results:

1/20 depot lepromin used only

40 out of 45 converted; 1 did not; 3 were indefinite; 1 could not be read because he developed extensive scabies.

1/100 depot lepromin used only

15 out of 16 converted; 1 did not.

Both lepromins used

9 out of 13 showed conversion in both; 1 did not; 3 went away.

Including the 9 children referred to above, where the interval was five weeks, at least 71 out of 83 children were shown to have converted by the depot tests. The results with the 1/100 depot lepromin were not greatly different from those with the stronger preparation. The proportion in which conversion was known to have been indicated is similar to that reported by other workers when intradermal lepromin testing with 1/20 antigen was done both before and after vaccination (Kinnear Brown et al. 1957). A strength of 1/50 may ultimately be preferred; but, where the number of tests can be reduced from two to one, the number of people that can be examined is doubled, with correspondingly less inconvenience to them.

Summary and Conclusions

A method of lepromin testing is described using the Heaf multipuncture apparatus. It is believed that by this method a 1/100 depot lepromin will identify the weak reactor. In the investigations reported depot lepromins remained in the skin long enough to indicate conversion after subsequent B.C.G. vaccination.

Depot lepromin is easier to make, and by this method it is easier to administer. The tremendous economy in material—especially if the number of tests can be reduced from two to one—will make it possible to examine the children and contacts of patients so as to identify those most likely to develop the disease. This should open the way to further investigations of the value of B.C.G. vaccination in protecting children against leprosy.

The test produces neither ulceration nor scars nor any other unpleasant sequelæ—important considerations to those it is desired to examine.

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PROPHYLACTIC TRACHEOTOMY IN THE TREATMENT OF CARDIORESPIRATORY DISEASE ASSOCIATED WITH SURGICAL CONDITIONS

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OPERATIONS are nowadays done on patients—often elderly—whose illness is complicated by respiratory and/or cardiac disease. In some cases there is time to treat the complication before operation, but its response may be unsatisfactory; in others the time cannot be spared owing to the urgency of the surgical condition.

This paper is concerned mainly with those patients who come to operation with severe bronchospasm, bronchitis and emphysema, and a liability to cor pulmonale. But similar principles apply to patients with severe mitral stenosis who are likely to develop copious watery secretions after operation. Until recently cardiorespiratory disease has been treated after operation along conservative lines. Bronchoscopic aspiration was used only occasionally, and tracheotomy, if used at all, was often put off until too late.

Tracheotomy benefits several pulmonary and cardiac conditions, by the following means (Reynolds et al. 1950, Pitman and Wilson 1955, Williams 1955, Walford 1955):

(1) Decreasing the resistance to respiration.

(2) Diminishing the dead space, and thereby allowing increased alveolar ventilation with less work.

(3) Providing an easy way of clearing secretions from the larger air-passages by mechanical aspiration.

(4) Allowing prolonged artificial respiration when necessary.

(5) The use of a cuffed tube, which prevents aspiration of regurgitated fluid.

We suggest that tracheotomy should be performed more often upon such patients, and much earlier. Sometimes the likelihood of severe postoperative cardiorespiratory trouble is so evident beforehand that prophylactic tracheotomy, immediately after operation, is a sensible precaution and avoids an unpleasant second operation.

The following cases are described as examples of common cardiorespiratory diseases complicating general surgical conditions. They illustrate the degree of severity for which prophylactic tracheotomy is advisable.

Case-reports

Case 1.—A man, aged 73, was admitted to hospital complaining of 24 hours' general abdominal pain and vomiting. He had had bronchitis and emphysema for 40 years.

He was orthopnœic, with noisy laboured breathing, copious yellow sputum, and mild cyanosis. (Temperature 101·2°F, pulse 100, respiration 34.) His chest was fixed, with crepitations at the left base and widespread rhonchi. There was no evidence of cardiac failure. His abdomen showed the signs of widespread peritonitis.

Although an experienced physician thought that the state of his chest precluded operation, he was given an anæsthetic, tracheobronchial aspiration was carried out, and a ruptured appendix was removed.

Tracheotomy was performed immediately after closing the abdomen. His respiration was then managed in the usual way with a humidifier, intermittent tracheobronchial aspiration, breathing exercises, antibiotics, and bronchodilators. 7 days after operation his tube was removed, and he was discharged on the 29th day.

Case 2.—A man of 76 was in hospital for another condition when a peptic ulcer perforated. He had a history of chronic ill health, having been admitted from time to time for bronchitis, emphysema, hypertension, and cardiac failure. He was having maintenance doses of digitalis and diuretics.

He was orthopnœic, with cyanosis, clubbing, and much sputum. His chest was in a similar state to that of the first patient, with the additional signs of mild right-sided cardiac failure.

A prophylactic tracheotomy was done immediately after closing the abdomen. The tube was removed after 7 days, and he was discharged from hospital on the 17th day.

Case 3.—A man of 55 was admitted to hospital having had 4 months' dysphagia from carcinoma of the lower end of the œsophagus. He had had chronic bronchitis for 30 years, and attacks of pneumonia in 1954 and 1956.

He was orthopnœic, with finger clubbing and a productive cough, and his chest was in a similar state to that of the first patient. Radiographs showed bilateral apical shadowing, with possible tuberculous infiltration in the left mid zone. No acid-fast bacilli were found in the sputum.

Despite preoperative therapy, there was little improvement in his chest 15 days later, when the lower end of his œsophagus was resected through a left thoracoabdominal incision. The postoperative management of his chest was along the lines indicated above. The tracheotomy tube was removed after 7 days, and progress was uneventful.

Case 4.—A man, aged 62, had had mild bronchitis and emphysema for 5 years. In the last 6 months it had become rapidly worse, and he had developed cor pulmonale.

He was admitted for the cure of an epigastric incisional hernia which had followed the emergency resection of a ruptured abdominal aortic aneurysm by Prof. J. B. Kinmonth 2 years previously. This hernia interfered with his very limited power of coughing, and it was painful.

On examination his respiratory system was worse than that of the previous patients. Radiographs showed infiltration of the right upper zone, and his sputum contained acid-fast bacilli. He was transferred to the medical side for preoperative therapy, but improvement was slight, and his serum-bicarbonate level remained high.

Prophylactic tracheotomy was advised immediately after operation (by Professor Kinmonth) a month later. In addition to the other indications, we thought he might be unable to breathe adequately by himself because diaphragmatic movement would be limited by the mechanical effects of the herniorrhaphy and by incisional pain. This was so, and it was necessary to put him on a respirator, which was used continuously for the first few days. He was gradually weaned off it, but his breathing was not satisfactorily restored for 2 weeks.

Discussion

The importance of performing tracheotomy early if at all is now being realised. It is a minor procedure, which is highly successful not only in preventing worsening of the cardiorespiratory state but also in improving upon the preoperative condition. If tracheotomy is likely to be necessary after operation, prophylactic tracheotomy is suggested so that the important interval of the first few postoperative days without tracheotomy is avoided, and a second operation, often under difficult circumstances, is not needed.

Unfortunately tracheotomy is still too often left until the patient is exhausted by repeated efforts at coughing, and the complications which should have been prevented have developed. Many examples could be given of the futility of performing tracheotomy too late. This lack of " success ", combined with a dread of the supposedly mutilating procedure, has in the past helped to deny its use. In fact patients accept it well and soon become adjusted both mentally and physically. They are able to cooperate with their treatment and are safer at night, making their nursing much easier.

Complications of tracheotomy should not occur if the tube is kept in place for only a short time, provided the correct technique and the correct tube are used, and the staff are trained in managing such patients. Additional difficulties arise when a respirator has to be used. The only disadvantage we have encountered is delay in healing when much purulent sputum is still being produced, and this has necessitated late suture of the wound in some cases.

Summary

For patients undergoing operation who have severe cardiorespiratory disease early tracheotomy can be valuable.

When possible, tracheotomy should be performed immediately after the main operation.

We should like to thank the surgeons under whose care these patients were treated for permission to quote their histories; and Dr. M. D. Nosworthy and Mr. N. R. Barrett for help in preparing this paper.

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Reviews of Books

A System of Ophthalmology

Vol. 1. *The Eye in Evolution.* Editor: Sir STEWART DUKE-ELDER, G.C.V.O., LL.D., M.D., F.R.C.S. London: Henry Kimpton. 1958. Pp. 843. 126*s*.

Duke-Elder's classic *Textbook of Ophthalmology* has already become a universal reference book, and the first four of its seven volumes are due for revision, having been out of print for some time. It will reappear in the new and expanded form of *A System of Ophthalmology*, under joint authorship in fifteen volumes, of which Sir Stewart has himself undertaken the first.

Its scope extends considerably beyond the limits of clinical ophthalmology: it embraces the development of visual function from the early appearance of photosensitive cells in primitive organisms, through the stages in which more highly organised receptors appear, to the ultimate appearance of the complex faculties of vision and apperception found in primates. The book is concerned first, therefore, with the effects of light upon living organisms, subsequently with the evolution of the visual apparatus itself, and, in later chapters, with that of the increasingly accurate and perceptive functions of the eye in higher animals. Thus, its interest will primarily be for the biologist; but the serious student of ophthalmology will find here a fascinating account of the evolutionary background to his subject, and the ophthalmologist engaged in experimental animal work a valuable compendium of comparative anatomy and physiology.

The book is written with the author's customary facility, the text enlivened with original marginal drawings, and profusely illustrated with photographs and diagrams. It goes far to convey the fascination which the subject evidently has for its author, and, though its contents will hardly be regarded as obligatory reading for the ophthalmological graduate, he will gain from it a stimulating perspective of his subject, which cannot have its equal in any specialty.

The Effect of Advancing Age Upon the Human Spinal Cord

L. RAYMOND MORRISON, M.D., with the collaboration of STANLEY COBB, M.D., WALTER BAUER, M.D. Cambridge, Mass.: Harvard University Press. London: Oxford University Press. 1959. Pp. 127. 48*s*.

IN all branches of pathology, but perhaps most of all in neuropathology, a knowledge of the normal is essential. This book, written by a distinguished neuropathologist who died some years ago and edited by his colleagues, is designed to illustrate the "normal" changes which occur in the human spinal cord with advancing age. Cords from patients ranging in age from the second to the ninth decade were examined histologically by Dr. Morrison, who used the routine neurological stains for studying the various elements. Changes affecting the nerve-cells (and less noticeably the myelin, glia, axons, and blood-vessels) were observed consistently from the fourth decade onwards, and as might be expected tended to increase throughout life, though some cases which from their age would have been expected to show fairly severe changes did not do so.

The deviations from the normal found in 31 cases are described and illustrated in the first part of the book, and there follows an atlas, with descriptive text, showing the configuration of each segment of a normal spinal cord and the numbers of anterior horn cells counted at each level. Since Bruce's classical *Atlas of the Spinal Cord* has long been out of print, this atlas will be a welcome addition to the library of all neuropathologists.

None of the spinal cords used in this study came from a strictly "normal" case since the patients had all died of diseases such as malignant hypertension, cancer of the stomach, and miliary tuberculosis (even the cord on which the atlas is based came from a young woman with subacute bacterial endocarditis and showed at least one focus of acute degeneration, presumably embolic). It seems unfortunate that patients with such diseases, which may well affect the spinal cord directly, were studied; but the authors felt that it was of value to use what they call run-of-the-mill spinal cords.

Nevertheless, this is a very useful study of a subject about which little is known, and it points the way to much further research work which must be done before we have any very complete knowledge of age changes in the human spinal cord. The book will be widely used by those studying the nervous system.

An Atlas of Surgery

F. WILSON HARLOW, M.B., F.R.C.S., consultant surgeon, South West Metropolitan Regional Hospital Board. London: Heinemann Medical Books. 1958. Pp. 374. 50*s*.

Atlas of Technics in Surgery

Editor: JOHN L. MADDEN, M.D., F.A.C.S., assistant clinical professor of surgery, New York Medical College. New York: Appleton-Century-Crofts. London: Mayflower Publishing Co. 1958. Pp. 648. £12 12*s*.

THERE is little to say about atlases: their very intention is to dispense with the written word. *An Atlas of Surgery* depicts the field a student of today is expected to cover in surgery. Clearly it accepts the status quo: that medical students are an overworked race, since it is necessary to pour so many facts into their minds. In a brief foreword Sir Cecil Wakeley extols the value of the picture against the written word: the picture makes a stable imprint on the memory. But might not the imprint be too stable? Is that not a fault of our present curricula, that they induce stability, not adaptability? This is instruction, not education. As a ruthless imparter of facts this atlas excels, but it makes no great appeal to the intelligence.

For the *Atlas of Technics in Surgery* there is more to be said, not only because it has more words, but because the purely operative aspect of surgery can with no stretch of the imagination be considered a field of education; its learning is a vocational chore to be withheld from the student and undertaken only as an obvious necessity by the intending surgeon. An emphasis on illustrative methods is excusable under these circumstances and it is a pleasure to record that the standard of illustration on which this book so heavily relies is more artistic than in many recent works, though a few of the drawings suggest that medical illustration could profitably return to the standards of Cheselden and Calcar. The contents are hugger-mugger in presentation but within one volume the editor, assisted by 64 renowned contributors, has contrived to present most of the operations outside the special specialties (such as neurosurgery, otorhinolaryngology, urology, and plastic surgery) and provide useful references.

Psychiatry in Medical Practice

Editor: W. LINDESAY NEUSTATTER, M.D., B.SC., M.R.C.P. London: Staples Press. 1959. Pp. 311. 42*s*.

LIKE Dr. Neustatter's earlier book, *Modern Psychiatry in Practice*, this is a simplified guide or textbook, designed mainly for general practitioners. Compared with its predecessor, the style is less racy, and the statements more cautious, but readers are left in no doubt as to the author's standpoint. Chapter XIV, for example, on the psychiatric examination and treatment of children, provides convincing evidence of his tart dislike of jargon and empty pretensions; throughout the book the same downrightness is conspicuous. Based as it is on much experience, this work (to which four of Dr. Neustatter's colleagues have also contributed) gives practitioners and students a balanced, moderate, and readable picture of what the common psychiatric disorders are like and how they are nowadays treated.

A Practice of Thoracic Surgery (2nd ed. London: Arnold. 1958. Pp. 619. 100*s*.).—It is only just over five years since this work by Mr. A. L. d'Abreu made its début; and the need for another edition of so specialised a book reflects its success. It must in part be due to the dissemination of knowledge of thoracic surgery by such books as this that the subject has moved far enough in five years to necessitate considerable revision in the sections on cardiac surgery and pulmonary tuberculosis.

THE LANCET
LONDON 20 JUNE 1959

Inapparent and Subclinical Pyelonephritis

WHILE it is being increasingly recognised that pyelonephritis is the commonest cause of chronic nephritis and therefore of secondary hypertension, there is still a considerable discrepancy between the number of cases detected post mortem and the number diagnosed clinically.[1–3] In investigating this discrepancy, several groups of workers have undertaken follow-up surveys of patients who had had clinical episodes of acute pyelonephritis, not only among children as was strongly recommended by PORTER and GILES [4 5] but also in adults. MACAULAY and SUTTON [6] found that of 32 children followed up six or seven years after admission to hospital on account of urinary infection, 5 had died with gross organic deformities of the renal tract, 13 had either recurrent or persistent signs of urinary infection, and only 13 were apparently normal (1 was untraced). These workers cite as the best follow-up survey that of WOODRUFF and EVERETT,[7] who recalled 76 female patients who had had childhood pyuria between five and twenty-three years before: 22% had had only the one clinical episode but showed an abnormality of the renal tract at the follow-up, 29% had had repeated attacks of urinary infection, and only 49% had had only the one attack and showed no evidence of renal disease at the follow-up. These results indicate that pyuria in infancy and childhood is not a simple and easily controlled condition, and emphasise its sinister potentialities in leading to a chronic disorder with its complications. In adults the position is apparently not so bad. DOCK and GUZE [8] investigated 30 patients (all female) ten weeks to five years after an attack of acute pyelonephritis and found that 8 patients, 5 of them completely symptomless, continued to excrete organisms (in 6 cases the same organism that had caused the original episode) some time after the acute episode but none for longer than eighteen months. KASS [9] found significant bacilluria persisting for slightly longer periods: also that symptomless bacilluria, found by chance, might suddenly develop, weeks or months later, into a typical attack of acute pyelonephritis. In the large series of GARROD et al.,[10] who were investigating the results of chemotherapy in urinary infections, 15 out of 137 patients with no history of previous infection and no urinary tract abnormality gave a positive urine culture two to four months after treatment of the primary acute episode, while 34 out of 95 patients with a history of previous infection gave a positive culture at a similar time after treatment of the episode: of 85 patients with urinary tract abnormalities, 63 gave positive cultures two to four months after treatment.

In male patients a carefully taken mid-stream specimen of urine has long been accepted for bacteriological investigation; but convention has demanded a catheter specimen from the female. One of the difficulties facing investigators of pyelonephritis, especially in these follow-up surveys, has been the fear of causing fresh infection or spread of existing infection by catheterisation: it is strongly hinted [8] that one reason for the common occurrence of pyelonephritis in pregnancy is the custom of passing catheters. To avoid this danger, SANFORD [11] and KASS [12] have been using with considerable success a mid-stream specimen from women, too, followed by a quantitative estimation of the bacteria present. The specimen must be examined within one hour of collection or else frozen until examined. Most specimens contaminated during collection contain less than 1000 bacteria per ml. of urine, whereas in typical acute pyelonephritis specimens contain more than 100,000 bacteria per ml. KASS [9] found that only 1% of specimens fell within the doubtful range of 10,000 to 100,000 per ml. A fairly good correlation was found to exist between the concentration of bacteria present in the urine and their recognition on microscopic examination in stained smears: SANFORD found that for bacteria to be seen on stained smears not less than 5000–10,000 per ml. must be present, while KASS [9] reported that staining for bacteria was about 80% reliable. He suggested [9] that estimation of the number of organisms is a more reliable guide than examination for pyuria, since pus cells may be present in some specimens but absent from others.

SANFORD points out that pyelonephritis, while clinically apparent as a series of separate and seemingly unrelated events, should be regarded as a disease continuum; and he modifies the hypothetical illustration from JAWETZ [13] where the level of symptoms may be overstepped in infancy, in "honeymoon cystitis", in "pyelitis of pregnancy" and in acute episodes of pyelonephritis, while the baseline of healing may be reached after such episodes; there is, says SANFORD, a zone between symptoms and healing involving "inapparent active" or "inactive" stages during which the disease may continue, leading to symptomless bacteriuria and eventually to uræmia or hypertensive heart-failure. Recognition of this "inapparent active" stage, in which vague and very indefinite symptoms may be shown on urinary examination to be due to an otherwise unsuspected pyelonephritis, may lead to thorough treatment and thus stop the otherwise insidious course.

This conception of "inapparent activity" does not completely account for the discrepancy between the frequent postmortem finding of pyelonephritis and the small numbers with a clinical history of the disease. Perhaps further instances would be brought to light in

1. *Lancet*, 1956, i, 1000.
2. Jackson, G. C., Dalienbach, F. D., Kipnis, G. P. *Med. Clin. N. Amer.* 1955, **39**, 297.
3. MacDonald, R. A., Levitin, H., Mallory, G. K., Kass, E. H. *New Engl. J. Med.* 1957, **256**, 915.
4. Porter, K. A., Giles, H. McC. *Arch. Dis. Childh.* 1956, **31**, 303.
5. *Lancet*, 1956, ii, 1036.
6. Macauley, D., Sutton, R. N. P. *ibid.* 1957, ii, 1318.
7. Woodruff, J. D., Everett, H. S. *Amer. J. Obstet. Gynec.* 1954, **68**, 798.
8. Dock, D. S., Guze, L. B. *Ann. intern. Med.* 1959, **50**, 936.
9. Kass, E. H. *Trans. Ass. Amer. Phycns*, 1956, **69**, 56.
10. Garrod, L. P., Shooter, R. A., Curwen, M. P. *Brit. med. J.* 1954, ii, 1003.
11. Sanford, J. P. *J. Amer. med. Ass.* 1959, **169**, 1711.
12. Kass, E. H. *Arch. intern. Med.* 1957, **100**, 709.
13. Jawetz, E. *Disease-a-Month*, November, 1954.

life if patients with vague symptoms of backache and temperature (which are often put down to mild influenza or other indefinite diagnosis) had their urine investigated: these ill-defined illnesses may well be episodes of "subclinical" pyelonephritis requiring vigorous treatment.

Annotations

PRINTING OF THE LANCET

OWING to the continuing dispute in the printing trade, the present issue of the journal is severely curtailed. If meanwhile no settlement has been reached, normal printing may be completely interrupted at the end of this week. In that event we shall keep in touch with readers until normal issues can be resumed.

MYCOBACTERIAL PSEUDOTUBERCULOSIS

IN the past few years illness simulating tuberculosis has been attributed to "atypical" mycobacteria with increasing frequency.[1] More careful appraisal both of acid-fast bacilli formerly dismissed as contaminants and of those accepted as tubercle bacilli has disclosed a considerable number of infections for which it seems legitimate to adopt the name "pseudotuberculosis". In Wales Marks [2] found that the incidence of bacteriologically proved cases of pseudotuberculosis was a little over 1% of that of open tuberculosis.[2] About half the pseudotuberculosis cases were associated with pneumoconiosis; and probably other lung diseases, including quiescent tuberculosis, also predispose to the infection. Men are chiefly affected.

The diagnosis of mycobacterial pseudotuberculosis is usually an unexpected outcome of bacteriological examination; for the clinical, radiographic, and, in most cases, histological appearances are those of tuberculosis. The "atypical" mycobacteria responsible are a somewhat heterogeneous group distinguished with certainty only by their association with the disease. In practice the diagnosis may be made fairly confidently when a lesion repeatedly yields a strain of mycobacteria distinct from tubercle bacilli, provided care has been taken to exclude commensal and contaminating mycobacteria during the collection and culture of the material. A single isolation has little or no significance. Tubercle bacilli are very occasionally masked in culture by other mycobacteria present in the same specimen. It is therefore advisable before making a firm diagnosis of pseudotuberculosis to inoculate a guineapig with the culture and another directly with suitably treated sputum or other morbid material. To the properties of "atypical" mycobacteria [1] may now be added the positive reaction for arylsulphatase given by almost all strains [3] and the negative reaction for niacin.[4] The latter distinguishes the group from human-type tubercle bacilli of low virulence.

Patients with mycobacterial pseudotuberculosis and tuberculosis are commonly housed together. This is undesirable since cross-infection in either direction, though it has not yet been described, remains a possibility. The infectivity of pseudotuberculosis for contacts in normal health is probably negligible. "Atypical" mycobacteria are resistant to many of the drugs used to treat tuberculosis. Fortunately the lesions are often of limited extent, and if chemotherapy fails their excision may be possible.

The habitat of "atypical" mycobacteria is unknown. They may be free-living organisms which in favourable circumstances can survive and multiply after accidental access to human tissue. It is very improbable—although perhaps not impossible [5]—that they are degraded tubercle bacilli. It would be tempting to relate any true increase in mycobacterial pseudotuberculosis to present trends in tuberculosis. People with a poor defence against tubercle bacilli now commonly survive, either with the help of chemotherapy or by escaping infection altogether. Are they potential victims of pseudotuberculosis? There is much to learn about this disease, and the formation of a working party of the Public Health Laboratory Service to study it is timely. One urgent need is for agreed names both for the infection and the organisms responsible.

SEQUEL OF MYOCARDIAL INFARCTION

FOUR years ago Dressler [6] first drew attention to an unusual complication of myocardial infarction characterised by pleuropericarditis.[7] Subsequently Dressler and his colleagues published a further report,[8] and he has recently described his experiences with 44 cases of what he now calls the "postmyocardial-infarction syndrome".[9] A pericardial rub is not uncommon when necrosis involves the epicardial surface of the heart, but it is short-lived and often passes undetected unless it is specifically sought by daily auscultation after the initial infarction. Detectable effusion is rare, though it was found in 2% of Pearson's cases.[10] By contrast, pericarditis with a conspicuous rub and with appreciable effusion is a main feature of the postmyocardial-infarction syndrome. It may develop immediately, or appear as long as the eleventh week after infarction, and shows a remarkable tendency to recur when all seems set for recovery. Severe pain is almost invariable, resembling that of infarction itself in character and site, though differing in its accentuation by deep breathing, yawning, movement in bed, and sometimes swallowing. Fever, often long-continued and intermittent, accompanies the pain. Pericardial effusion, judged radiographically by changes in heart size, occurred in 62% of Dressler's cases, and small pleural effusions on one or both sides were even commoner. 6 patients had pleural effusions which required tapping; in 3 of these cases the effusions were heavily bloodstained. In more than a quarter of patients patchy consolidation at the lung bases was observed, and this was unexplained by cardiac failure, infection, or embolism.

Correct diagnosis depends on awareness of the syndrome, and suspicion should be aroused by the unusual persistence of fever or its reappearance, or by recrudescence of pain, which may immediately suggest fresh infarction, though the electrocardiogram remains unchanged or indicates pericarditis. Enlargement of the heart shadow, with pleural effusions or patchy consolidation at the bases, clinches the diagnosis if other causes can be excluded. The syndrome is less likely to cause confusion when it develops late or when it recurs. Bouts may

1. See *Lancet*, 1955, ii, 758.
2. Marks, J. *Brit. J. clin. Practice*, 1958, **12**, 763.
3. Whitehead, J. E. M., Wildy, P., Engbaek, H. C. *J. Path. Bact.* 1953, **65**, 451.
4. Konno, K., Kurzmann, R., Bird, K. T., Sbarra, A. *Amer. Rev. Tuberc.* 1958, **77**, 669.
5. Tarshis, M. S. *ibid.* 1958, **78**, 921.
6. Dressler, W. *Circulation*, 1955, **12**, 697; *J. Amer. med. Ass.* 1956, **160**, 1379.
7. See *Lancet*, 1956, ii, 79.
8. Dressler, W., Yurkofsky, J., Starr, M. C. *Amer. Heart J.* 1957, **54**, 42.
9. Dressler, W. *Ann. intern. Med.* 1959, **103**, 28.
10. Pearson, H. E. S. *Brit. med. J.* 1953, ii, 4.

continue for as long as three months, sometimes necessitating readmission to hospital: Dressler observed relapses after ten and twelve months in 2 patients. Early diagnosis is important so that the patient and his relatives may be reassured about the outcome: in Dressler's series only 1 patient died; and this death was from hæmopericardium, which was attributed to the continuation of anticoagulants after the onset of pericardial involvement. The dangers of anticoagulants in the presence of generalised pericarditis have been emphasised by Goldstein and Wolff,[11] and such treatment should be withheld or stopped when the post-myocardial-infarction syndrome is suspected. Rest and analgesics are sufficient in most cases; but adrenal steroids, which rapidly relieve pain and fever, may be required when the disorder runs a relapsing course.

The cause of this syndrome remains unknown: it follows mild as well as massive infarction; and it is independent of anticoagulant therapy, which seems to exclude leakage of blood into the pericardium as the exciting factor. No infective agent has been so far incriminated, and antibiotics do not affect its course. Similarity to the post-commissurotomy syndrome, and also to the ill-defined entity of " benign idiopathic pericarditis " has been emphasised by Dressler, who suggests that all these result from sensitisation to an antigen produced in susceptible individuals by myocardial necrosis. This hypothesis is based on the experimental production of allergic pericarditis and pneumonitis in animals,[12] and the occurrence of pericarditis in allergic states in man.[13] It would be interesting to know whether victims of the syndrome are liable to recurrence of it after further infarction at a later date. Whatever its true nature, Dressler's clear account will enable the condition to be recognised by many who in the past must have been puzzled by its manifestations.

MOTHER'S ROLE IN INFECTIONS OF NEWBORN

MOST infants are bacteriologically sterile at birth, and the first bacteria they acquire are likely to come from the mother. Their earliest flora is said to resemble that of the mother's vagina,[14] and after the first feed organisms characteristic of her mouth are added [15 16]; few of these seem to do any harm. But infants born in hospital are subjected to a more severe bacteriological hazard—invasion by the " hospital bacteria ". Nowadays *Staphylococcus aureus* is the most important of these organisms. Attempts have been made to ascribe its introduction and spread to the mothers, but phage-typing has shown that they are rarely responsible. *Streptococcus pyogenes* is no longer the scourge that it used to be in maternity hospitals, but it still causes disquieting incidents. New light was thrown on its epidemiology in 1956 by Boissard and Eton [17] and Kwantes and James,[18] who showed that the umbilical stump of apparently healthy infants is an important reservoir. Pollard and Perry [19] confirm these findings. Although mothers, like nurses, may bring this organism into the hospital, its spread is due to other factors.

A third organism which can cause " hospital infection " among newborn babies is *Escherichia coli*—or rather certain serotypes which are known to be associated with infantile enteritis. The importance of the mother in the epidemiology of this disease has been investigated by Cooper et al.[20] Rectal and lochial swabs were taken from 361 women at the time of their confinement, and 46 (12·7%) were found to be positive for these types of *Esch. coli*. 40% of these mothers infected their babies with a similar strain within four days of birth. There was little evidence of cross-infection with these strains, and of 114 members of staff only 2 carried them. This is good evidence that mothers carrying certain bowel organisms commonly transmit them to their babies.

Although 46 mothers were infected with pathogenic *Esch. coli*, only 1 mother and 1 infant developed diarrhœa. On the other hand, of 40 infants who were negative and whose mothers were negative while in hospital, 28 developed diarrhœa at home in the first 3 months of life; from 11 of these, pathogenic *Esch. coli* were isolated. Cooper et al. suggest that the first group of infants might have been protected by maternal antibodies, but there is little evidence that these are formed in any quantity or that there is cross-protection between somatic antigens. Possibly the difference may have been associated with the types of *Esch. coli* encountered. 80% of the strains isolated outside hospital were of the types which have been associated with epidemics, compared with 50% of those in hospital. The older children in a family may be a more dangerous source of virulent strains than mothers in hospital. A mother would be dangerous if she carried a virulent epidemic strain and gave it to the newborn baby.

We have much to learn about the different levels of virulence of these organisms; but, once a highly virulent strain enters a nursery, it is almost impossible to prevent its spread. Should we, then, start screening the bowel organisms of women before admitting them to maternity hospitals? It would be difficult to place any trust in the results until a reliable selective medium for pathogenic *Esch. coli* has been found.

BIRTHDAY HONOURS

ON his election in 1957 as president of the Royal College of Physicians, Prof. Robert Platt was already known in and beyond Manchester as a physician, the head of a lively and happy department, a thinker, and a musician respected by professionals; and he has subsequently added to his stature by his leadership of the college in a time of change. The baronetcy conferred on him in the Birthday Honours, announced last week, will be applauded. The appointment of Sir Archibald Gray and Dr. Wilfrid Sheldon as K.C.V.O. and of Air Vice-Marshal F. W. P. Dixon as K.B.E. will also be widely welcomed. Prof. Dugald Baird, of Aberdeen, who is created a knight, has made a mark both by his teaching and by his notable work on obstetrics in its social setting. Prof. A. J. Lewis, who also receives a knighthood, has contributed signally to psychiatry by applying to it an incisive and critical mind, objective judgment, and lucid exposition. Those in the Commonwealth on whom knighthood is conferred include Mr. Alexander Gilles, of Wellington, in recognition of his contributions to orthopædics. We congratulate these and other doctors named in the Honours List; and we regret that, owing to printing difficulties, we cannot at present name them all.

11. Goldstein, R., Wolff, L. *J. Amer. med. Ass.* 1951, **146**, 616.
12. Gregory, J. E., Rich, A. R. *Bull. Johns Hopk. Hosp.* 1946, **78**, 1.
13. Ellis, R. V., McKinlay, C. A. *J. Lab. clin. Med.* 1941, **26**, 1427.
14. Witkowski, R. *Zbl. Bakt.* 1935, **133**, 334.
15. Gundel, M., Schwarz, F. K. T. *Z. Hyg. InfektKr.* 1932, **113**, 411.
16. Torrey, J. C., Reese, M. K. *A.M.A. J. Dis. Child.* 1945, **69**, 208.
17. Boissard, J. M., Eton, B. *Brit. med. J.* 1956, ii, 574.
18. Kwantes, W., James, J. R. E. *ibid.* p. 576.
19. Pollard, B. R., Perry, S. E. *Mon. Bull. Minist. Hlth Lab. Serv.* 1959, **18**, 46.
20. Cooper, M. L., Keller, H. M., Walters, E. W., Partin, J. C., Boye, D. E. *A.M.A. J. Dis. Child.* 1959, **97**, 255.

Special Articles

EMERGENCIES AT SEA

JAMES S. HALL
O.B.E., M.B. Lond., F.R.C.S.E.

CATASTROPHE comes quickly at sea. On any dark stormy night a doctor's bedside telephone may ring and the urgent voice of a coastguard may ask for immediate medical assistance to a ship several miles from shore. If he agrees, a lifeboat (R.N.L.I.) will be standing by to take him out. What should he take with him and what will he find when he gets there? Placing a doctor on board a ship in a heavy sea subjects a lifeboat crew to considerable danger; and the doctor must try to make this a risk worth taking.

THE EMERGENCY BAG

Doyen once remarked that the successful surgeon arose from a mountain of corpses. One might claim, less dramatically, that a successfully packed emergency bag can arise from some mountainous seas. Certainly, once the lifeboat is launched, there is no going back.

One needs a strong bag, the older the better. This applies also to clothes: on a stormy night we shall be lucky not to have both well soaked with oil and sea-water and battered against the ship's side before we return. When packing this bag it is wise to assume that no individual item, except plenty of unsterile water and the patient, will be found at the end of the journey. I have expected to use a well-equipped ship's hospital only to find it flooded and sliced in half after collision with the sharp prow of another vessel. I have asked for surgical spirit on an American ship only to find that every drop of alcohol on board, including the contents of gyroscopic compasses and ten jars containing pathological specimens, had been drained away by a dipsomaniac on board.

It is as well to be selfcontained for dressings, suture material, and minor instruments. I found that four 3 in. plaster bandages, two 3 in. rolls of 'Elastoplast', and a small supply of cellulose tissue sufficed for any fracture likely to be reduced on board. Morphine will probably have been given to any severely injured man but you must take your own anæsthetic. 'Thiopentone' and chloroform are the most convenient for single-handed emergency use. Although the mate will always offer to sit on the head of any obstreperous patient, the negligible risk of a small quantity of anæsthetic is much to be preferred.

It is advisable to pack things in some definite order in waterproof tins or boxes, which in turn can be labelled and wrapped in 'Cellophane'. Without this, plaster bandages and dressings will only survive one journey. Salt-water never seems to dry out, but the interior of the bag can be kept quite wholesome by putting a few paraformaldehyde tablets in the pockets or in loose gauze swabs.

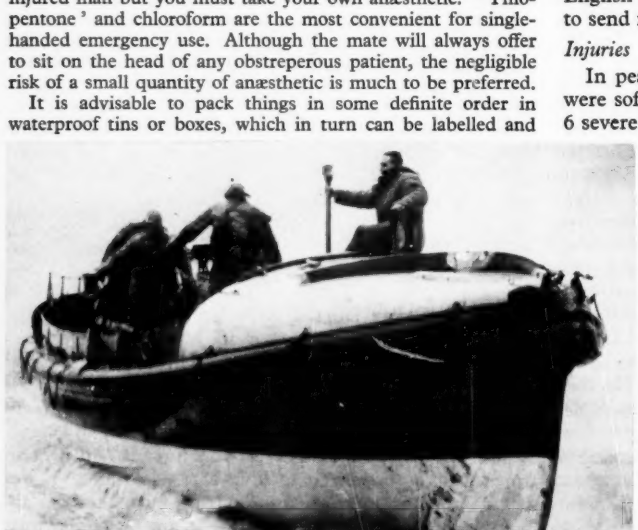


Fig. 1—Coming ashore in a 60-80 m.p.h. gale.

THE EMERGENCIES

Looking at my records I was surprised to find that the 100 cases seen at sea since 1946 differed little from the 700 I treated during the war.[1] In table I I have set out the emergencies which have caused 325 vessels in the English Channel, the North Sea, or the Downs, off Deal, to send messages asking for urgent medical help.

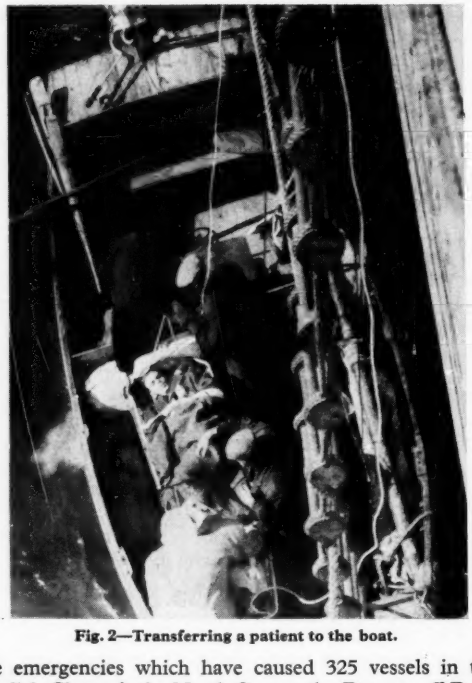


Fig. 2—Transferring a patient to the boat.

Injuries

In peace or war injuries head the list: of 84 cases 47 were soft-tissue injuries, 24 fractures, 7 dislocations, and 6 severe burns. Provision for these will take up half the contents of a sizable bag.

For a serious emergency it pays to take out your own stretcher or to borrow one before you go. The best type is the Neil-Robertson, which wraps round the patient and even enables him to be passed vertically through a manhole. The Americans have a deep-wire type with divided leg rests which is a good second. Probably the safest way of transferring a stretcher case to a small boat is to lower it down the ship's side with four separate ropes and a man holding each under the direction of a boatswain. As soon as the receiving party in the boat have a firm hold of the patient the ropes are dropped completely slack.

Abdominal Emergencies

Suspected appendicitis is a perpetual nightmare at sea and causes some 12% of all urgent calls. Diagnosis at sea is more accurate than on

1. Hall, J. S. *Brit. med. J.* 1946, i, 244

TABLE I—EMERGENCIES AT SEA WHICH HAVE PROMPTED 325 REQUESTS FOR URGENT MEDICAL HELP

Injuries	84	Renal colic	9
Suspected appendicitis	41	Foreign body in eye	8
Venereal diseases	35	Enteritis	8
Septic conditions	27	Intestinal obstruction	6
Pneumonia or bronchitis	17	Landed dead	6
Tonsillitis or sinusitis	17	Non-traumatic hæmorrhage	4
Infectious diseases	16	Acute rheumatism	4
Dental cases	12	Gastric or duodenal ulcer	4
Acute neuroses	11	Immersion	4
Heart-disease	10	Inoculations	2

shore, and I found over half the cases to be genuine. These are best landed as soon as possible: almost all of them required urgent operation.

The non-appendicitis cases fell into two groups. In the first the right-sided pain arose from conditions—such as gallstones, intestinal colic, abdominal strain, and direct injury—which often needed careful differential diagnosis. In the second group the pain was due to inguinal or scrotal conditions and required an examination a layman might hesitate to make. Among these I have seen an inguinal bubo, two hernias, acute hydroceles, several cases of acute orchitis, and a saphenous phlebitis.

Enteritis severe enough to initiate an emergency call was usually food-poisoning. This is commoner in small ships with no refrigeration facilities. A notable exception was a large American tanker which sent in a call for urgent medical assistance. On reaching the ship we were hailed by the purser: " Doc! Come up at once! I've got fifteen men dying of food poisoning. . . . Doc, you'll stay to lunch? " This was perhaps our most dangerous trip.

Intestinal obstruction varied from constipation to the real thing. To avoid repetition of an embarrassing experience it might be put on record that before exhibiting strong aperients and enemata to a Portuguese patient one should remember that in his language *a constipaçao* means a severe cold in the head.

Gastric and duodenal ulcers seem to be rare at sea and fortunately in twenty years I have encountered no perforation. The commonest causes of non-traumatic hæmorrhage have been epistaxis and bleeding piles. By the time one reaches the ship the trouble will usually have ceased and little more than examination and reassurance will be needed; but I believe that a few sod. calc. lactate tablets and liquid-paraffin emulsion respectively are more than placebos.

9 cases of renal colic were seen, and all had been diagnosed efficiently in advance. We landed 8 for further examination and treatment. A rubber catheter for acute retention should always be ready in the emergency bag.

Infections

10% of calls will be for some form of venereal disease—a dismal complaint which seems inseparable from medical practice at sea.

Twelve years ago it appeared that wholesale penicillin was about to obliterate spirochætes and gonococci, but this is far from being the case. Resistant urethritis and the sequelæ of partly treated venereal disease are all too common. I can only suggest that it is worth seeking the advice of the local venereal-diseases clinic. Most active cases on board will hope to be given an injection of benzathine penicillin and it is certainly worth putting a 10 ml. vial in the bag before visiting any ship.

Septic conditions are plentiful; and most abscesses are pointing by the time they become occasions for urgent calls. Ischiorectal abscesses are surprisingly common, and I could always open these easily with great relief to the patient. Some judgment is called for, however, in opening abscesses on board and I should now think twice before dealing with a quinsy. Unless the hæmorrhage is very slight, there will be frantic radio signals for further advice, or a firm request that the patient be landed for hospital treatment.

Fifth and sixth on the frequency list come the respiratory infections. Upper-respiratory infections, especially tonsillitis and sinusitis, have been four times commoner in American ships than in those of other nationalities.

It is tempting to blame the efficient central heating in American vessels: temperatures of 70°F are not unusual. One may also contrast the American habit of wearing in bed only the lower half of a pair of pyjamas, whereas Europeans prefer the upper half. Treatment of these cases must depend on the antibiotics available and the probable interval before further medical aid can be given.

I still remember the problem of a young Italian with pneumonia and a temperature of 105°F. The ship's next stop was South America and the only transport to the shore, seven miles away, was my small open boat half-full of snow.

Ordinary infectious disease on a ship is a great nuisance to everyone; luckily I have never had reason to suspect plague, smallpox, or cholera. Unless there are grave reasons for landing infectious patients it is better to leave them in their bunks, with the appropriate treatment. After all, so far as the ship is concerned, the damage has already been done.

Eyes, Ears, and Teeth

Perhaps because they are such a source of pain and anxiety at sea, foreign bodies in eyes seemed more common than appears in table I. A spud and a little 5% cocaine should be in the bag; and a pocket-torch and a magnifying-glass help. Ship lighting is seldom powerful, and the torch can be valuable in many non-medical ways. With an elementary knowledge of the Morse code we often sent useful signals across several miles of sea. A small Higginson's syringe was often useful for blocked ears.

Dental abscesses and severe toothache are common, and a pair of straight incisor forceps more than earn their place in the bag. In the daytime one can sometimes take a willing dentist, but I never managed to find one on a dark stormy night. Fortunately a little chloroform is a good substitute for a whiff of gas, and it never gave me any cause for anxiety.

Mental Illness

Cases of acute mental illness varied from nervous breakdown to homicidal mania. As most laymen have implicit faith in the power of a doctor to handle a dangerous lunatic, it is as well to ask a few questions before being introduced into the patient's cabin. Having had one narrow escape, I now look carefully to see whether any fire axes or dangerous implements are missing from their prominent places in the corridors. It is usually necessary to land these patients after making the necessary shore arrangements by radio-telephone.

Other Emergencies

I have found that " heart-disease " always means coronary thrombosis, and that the patient is usually the ship's captain himself. Fortunately all senior deck-officers hold a master's certificate; and more often than not, after reassurance, the mate (who prefers to be called the first officer) will decide to take the ship and its skipper to the home port. With rest and sedatives all our heart cases arrived safely.

Rheumatism as an emergency is likely to be some form of lumbago. It is a common ailment in any merchant vessel. Manipulation of a few recent cases of sacroiliac strain and occasional injection of 2% procaine into

TABLE II—NON-EMERGENCY AILMENTS FOR WHICH 845 PATIENTS RECEIVED MEDICAL ATTENTION ON BOARD 351 SHIPS

Asthma	1	Hydrocele	4
Appendicitis	25	Immersion	10
Found dead	6	Infectious diseases	16
Bronchitis	14	Influenza	33
Burns	12	Injuries	182
Cholangitis	12	Inoculations	92
Dental cases	25	Neurosis	24
Diabetes	1	Obstruction	6
Dislocations	7	Pneumonia	9
Enteritis	34	Pulmonary tuberculosis	7
Epilepsy	2	Renal colic	11
Epistaxis	3	Retention of urine	2
Foreign body in eye	12	Rheumatism	32
Fractures	27	Septic conditions	35
Gastric ulcer	3	Sinusitis	35
Gastritis	17	Skin disease	30
Hæmorrhoids	4	Tonsillitis	34
Heart-disease	13	Venereal diseases	59
Hernia	6		

localised fibrositis were my only ventures into the more dramatic types of treatment.

Immersion demands first-aid rather than medicine, though nikethamide and a flask of brandy or whisky are useful additions to the medical bag. The rum ration carried in lifeboats is deemed to be there for the resuscitation of the lifeboat crew rather than for the shipwrecked mariners whom they may collect, and I salute any doctor who has successfully broached it: every effort on my own part during the past twenty years has ended in failure. Although coxswains of lifeboats are pretty approachable fellows, there seem to be three stock replies to the question: " Coxswain, what about the rum ration? ":

1 (after four hours of drenching icy seas). " Sorry, Doc, the men ain't been exposed enough."

2 (later, when this obviously no longer applies). " Sorry, Doc, too rough to serve 'un."

3 (when conditions 1 and 2 and the weather make things possible). " Sorry, Doc, Summer Routine—no rum in summer." When one adds that the lifeboat summer seems to extend from March till November one can appreciate the difficulties ahead.

LESS URGENT PROBLEMS

Apart from the urgent cases, advice was often sought for other patients on board ship (table II). This list contains some surprises, among them the rarity of asthma and diabetes. Although adrenaline and insulin were almost the first drugs to be put in my seagoing bag, I never used either in twenty years. On the other hand, if one's favourite first-aid remedy for the remaining conditions is added to the contents of the bag, some very grateful patients will be left behind in almost every ship. One hopes, too, that the prompt production of an appropriate remedy may, in a small way, enhance the prestige of British medicine amongst the seafarers of many nations: in the freemasonry of the sea there is much wider publicity than ashore.

I often expected language difficulties but found few. American was the easiest language in which to get misunderstood. The word " aperient " will receive a blank stare from the purser; but " did he take physic? " obtains a prompt reply. And until " temperature " is changed to " fever " and " sick " to " vomit " there will be confusion of thought. But the standard of medical knowledge and emergency treatment is high. After an intensive course ashore, lasting a few months, the better pursers receive the interesting diploma of " pharmacist's mate ". After a further course they are permitted to give antibiotics.

NON-MEDICAL RECOMMENDATIONS

Lastly, some non-medical recommendations to my successors in this work.

It pays to follow all advice, however simple, offered by these boatmen. They know the anchorage and its dangers better than you do. Such hazards as there are occur in transferring the patients or yourself on or off a larger vessel. Even a small ship at anchor stays relatively still: small boats and lifeboats do not. Apart from the obvious advice not to release a firm handhold until you have found another, the keynote of safety is to let the ship's rope-ladder take you off the boat and vice versa. If you feel a lift under your foot you may safely transfer to either. To step off where you think the boat's gunwale is going to be is to invite disaster; and nobody who has dropped ten feet and sat on the boat's anchor will wish to repeat the experience. Also, once on the ship's rope-ladder, climb fast to make sure that if sixteen tons of lifeboat smash the rungs of the ladder, these rungs are the ones you have just left. I was also advised to make sure, when returning to a lifeboat, that the fattest man in the crew was directly underneath, but this scarcely adds to one's popularity the next time. But for my own part I am glad to say that so far I have been able to avoid implementing the special threat held over the heads of successive coxswains. We have not yet had a confinement on board their vessel.

I should like to pay a small tribute to the crew of the Walmer lifeboat for their many years of devoted service and the good care they have given to all my patients and myself. This has often been at the cost of long hours without food and successive nights without sleep. At times they have accepted considerable risk to ensure our safety, and on one occasion no fewer than five members of the lifeboat crew required treatment for injuries. For all this, they have received no special award other than the routine allowances authorised by the Royal National Lifeboat Institution.

Dialogues of Today

PROFESSIONS SUPPLEMENTARY

SOCRATES: I hope that your rheumatism is better, Apollodorus.

APOLLODORUS: Yes, thank you, Socrates, I have quite recovered, thanks to Eryximachus.

SOCRATES: What treatment did he give you?

APOLLODORUS: He didn't give me any, himself; but he said, " Go to Chloe, and get her to give you some physiotherapy ". So I did, and she did, with the result that you have heard. But here is Eryximachus. I was just telling Socrates, Eryximachus, how well Chloe has dealt with my rheumatism.

ERYXIMACHUS: Yes, she knows her job; but these girls are getting above themselves nowadays. In fact all the medical auxiliaries are. They want to speak of their work as their profession, and to control their training themselves, instead of leaving it to be controlled by the doctors, which is the only logical state of affairs.

SOCRATES: I am always glad to hear that logic is being applied to any practical matter, Eryximachus, so tell me why it is logical that the training of the medical auxiliaries should be controlled by doctors.

ERYXIMACHUS: Surely it is obvious, Socrates. What they do is a part of medicine. They use in diagnosis or treatment methods which have been devised by doctors, under the supervision of doctors, and to aid the purpose of doctors, which is to relieve, and if possible cure, the patient. It follows, surely, that the doctor must know better than the auxiliary how the things the auxiliary does should be done, and what training he or she needs in order to do them.

SOCRATES: Well, explain to me, Eryximachus, what a profession is, and why the term does not apply to the work of the auxiliaries.

ERYXIMACHUS: I find this a little difficult, Socrates, for the meaning of a profession seems to be getting somewhat blurred. Nevertheless I think it still has value. Surely a profession implies a number of things. Perhaps the essentials are certain aims and ideals beyond merely making a living, including the

service of the public, ethical standards, and a sense of pride in the calling itself, which unites those who belong to it.

SOCRATES: And is learning one of the attributes of a profession?

ERYXIMACHUS: Not essentially, I think; for although we speak of the learned professions there are others, for example, the armed services, which could hardly be called that.

SOCRATES: And is it necessary that a profession should govern itself?

ERYXIMACHUS: Certainly, for only in that way can it be sure of maintaining its ideals.

SOCRATES: Medicine, of course, is a profession.

ERYXIMACHUS: Of course!

SOCRATES: And you said just now that the work of the medical auxiliaries is a part of medicine. Are they not then part of the medical profession?

ERYXIMACHUS: I was speaking of medicine in a wider sense, Socrates. A profession requires a certain common background of ideas and training which doctors possess, but which medical auxiliaries do not. They may be part of medicine, but they cannot be part of the medical profession any more than a private soldier can be regarded as a professional man.

SOCRATES: Do you think that, as part of medicine, they ought to have the aims and ideals of the profession which you have mentioned—namely, a sense of service going beyond financial reward, ethical standards, and pride in their work?

ERYXIMACHUS: Certainly they should.

SOCRATES: Then would it not seem essential, if they are not to be part of the medical profession, that they should constitute their own professions having, as you rightly say, the same purpose as doctors?

ERYXIMACHUS: I don't mind their calling themselves the professions supplementary to medicine, if that gives them any satisfaction, but I still maintain that they should be controlled by doctors.

SOCRATES: But are the affairs of nurses controlled by doctors?

ERYXIMACHUS: That's different. Medical auxiliaries only do what doctors would otherwise have to do themselves, whereas nurses have many techniques of their own as well as what they do for doctors. In any case I'm not sure it wouldn't be better if doctors had more control over the training of nurses. As it is, in my opinion they are taught far too much medicine which they don't really need to know.

SOCRATES: You are indeed hard to please, Eryximachus; for, if I understand you aright, you think the training of the medical auxiliaries should be controlled by doctors because they are a part of medicine, and that of the nurses because they are not.

APOLLODORUS: You are an old humbug, Eryximachus, for you know perfectly well that when you sent me to Chloe you left the treatment to her. You wouldn't know how to turn on the apparatus, yourself, let alone what dose of the rays to give.

ERYXIMACHUS: That is beside the point, Apollodorus, which is that there are some doctors who are experts in all these matters.

ENTHUSIASTICUS: In my view, Socrates, Eryximachus is behind the times. Once again we are up against the speed with which medicine is moving. Doctors, let's face it, can no longer do everything in medicine. They've got to delegate much of their work to non-medical experts in order to get on with their own job, which is more and more to be the captain of a team. The question is whether the team will work better if it has some responsibility for what it does.

SOCRATES: Well, Eryximachus, it seems to me that we have agreed that the work of the medical auxiliaries is part of medicine, but that they are not part of the medical profession. You want them to maintain professional standards, and you say that a profession should govern itself. Does it not follow that the professions supplementary to medicine should govern themselves?

ERYXIMACHUS: You can always argue better than I can, Socrates, but all the same it wouldn't work!

Letters to the Editor

BOXING

SIR,—Reeling from the jolts of 24 medical references which end with Dr. Summerskill's Belgian uppercut, an amateur boxer of 30 years ago, such as myself, may well think twice before taking on medical evidence seemingly as solid as that quoted in your leading article of June 6. Nor do Messrs. Kaplan and Browder [1] (whose study of 1043 professional boxers had led them to "doubt the existence of the punch-drunk syndrome") lend much assistance, for my purpose is to defend not the professional but the amateur boxing game against what strikes as being a one-sided, loaded attack—an attack which must make countless fathers (who have held boxing, properly taught and properly controlled, to be a character-forming sport for boys and young men) wonder whether they have been conniving at some malpractice well within the lunatic fringe.

I should be the first to agree that, carried to excess, boxing must be harmful, though the degree of harm must vary with the degree of personal skill, for the boxer is taught that to guard himself is no less important than to score a point on his opponent, and that the head is by no means the only recommended target, as your leader suggests. I would go further and say that professionalism often leads to eventual injury—as often to the eyes as to the cerebrum—because the average professional seldom knows when to hang up his gloves: personal pride and inability to save are among the reasons.

The amateur is in a different category. Bouts are shorter than in the professional ring; they are better controlled and, generally speaking, the amateur's interest wanes after school and university and, more sharply, after marriage.

Where your leader is less objective than it could be is in the reliance it places on American data. So specialised has sport become in the U.S.A. that the amateur finds it necessary for success to develop a professional outlook in all he does—and when a game, particularly boxing, becomes a business rather than a sport it must tend to get out of hand.

Had a case prepared for a British readership been based on data drawn from this country, you would have more success in convincing those who, as myself, have boxed when young and have taught and judged boxing over the years, that the perils of boxing so far outweigh the positive virtues—courage, self-reliance, physical fitness, and chivalry—which the amateur game at the best teaches. Until that point is reached, I shall continue to advocate boxing as a perfectly proper sport for boys and young men so long as it is properly controlled and proper medical advice is available, but I personally think that it should be abandoned (as mostly it is) in the very early 20s.

Professionalism could be attacked and defended on quite different grounds, but that is a matter which does not concern my present argument.

IAN M. LESLIE
London, W.8. Vice-president, London Federation of Boys' Clubs.

BREAST CANCER

SIR,—I do not wish to add to the arguments for or against any specific type of treatment for breast cancer, nor am I competent to do so, but I venture with respect to point out the fallacy of one statement in your leading

1. *J. Amer. med. Ass.* 1954, **156**, 1138.

article of June 6 which reads: "Rosahn concludes that the various methods of treatment are either equally effective or exert no great influence on the disease; the former is probably the correct explanation since the improvement in successive quinquennial results over recent years seems most likely to be due to the earlier reference of patients for treatment: *if treatment had no influence the results should have remained the same*" (my italics).

This is clearly wrong. In order to dispose of some of the variables, suppose that the date of onset of breast cancer could be accurately determined and that all patients affected lived exactly 7 years irrespective of treatment. If they reported their disease in its 3rd year, the 5-year survival of cases after first being "treated" would be nil. If by propaganda they were persuaded to report the disease during its 2nd year, the 5-year survival from first "treatment" would be 100%. Earlier reference of cases must in fact mean longer survival from date of reference unless treatment is actively harmful.

The Royal Infirmary, Manchester. ROBERT PLATT.

SIR,—In your editorial of June 6 you give considerable thought to the difficulties of comparing survival-rates consequent on different methods of treatment for breast cancer, notably simple mastectomy and radiotherapy, versus radical mastectomy and radiotherapy. Neither in your editorial nor in any of the works referred to is consideration given to the biological activity of the breast tumours being treated. What proportion have an inherent tendency to grow and metastasise rapidly and what proportion are slow growing and metastasise late in their life-histories?

Of course, no-one can estimate these things by any reliable method. Histological examination is so often undependable even in the hands of the most experienced pathologist. The factor of biological activity might be thought to be equal in the two rival camps of Saskatchewan and Edinburgh. But is it? Saskatchewan, I believe, is one of the areas where cancer education is very enthusiastically pursued and routine check-ups on well women are carried out on a large scale—anyway on a far larger scale than anything of a similar nature in S.E. Scotland from which McWhirter's cases are drawn.

In such routine examinations of well women there are bound to be picked up a number of cancers which are either dormant or very slow growing and which left undisturbed might do their hosts no harm in ten or twenty years. No-one can say for certain how long. Naturally and correctly these cases are referred for surgery and might constitute 10% or 20% of the Saskatchewan cases and so account for the better survival-rate in that series.

It would indeed be enlightening if, in future statistical analysis of the survival-rates consequent on any particular treatment for breast cancer, there could be included a note of the number of breast cancers discovered in symptom-free women on routine investigation and the number discovered in the usual way by the patient themselves.

I suspect that the higher survival-rates emanating from some parts of the United States and Canada could be accounted for by the dormant cancers discovered by persons other than the patients themselves. These might include the husband as well as the cancer-detection clinic. Although not actually cancers-in-situ a large proportion of the tumours are probably slow developers and late metastasisers. Grouped and considered by themselves they might show a 90% survival-rate after any treatment provided it is not too drastic and lowering to the resistance. Are there any works which examine the survival-rates of cancers of the breast detected on routine examination only?

I do not think that stage-I cancers found in women who present themselves with suspected breast tumours already diagnosed are comparable with stage-I cancers found at detection clinics. In the former, in spite of what the patient says about accidental discovery while washing, &c., I am convinced that her attention is often drawn to the tumour because it is growing and making itself felt. It is biologically active and probably already metastasising, whereas the other may be dormant and only a potential danger.

Sheffield. E. C. ATKINSON.

RESOURCES FOR RESEARCH

SIR,—The comments in your leader [1] on this subject made many of us feel very sad over the situation. How much of this is due to your changed medical practice system, we do not know, but probably the relationship is quite direct. Please do not lose sight of the fact that most progress-ideas do originate in European minds and it is only our superior resources that enable us to "implement", as it were. You have stated it well.

One of our own great men, Harvey Cushing, was approached by a young postgraduate who had decided to go into research. Said he: "Can you tell me, Professor, what I must buy to equip my laboratory?" Cushing replied: "Running water, a basin, and some ideas."

Do not lose heart. We shall continue to need ideas.

Los Angeles. ARTHUR R. TIMME.

RUPTURE OF ANEURYSMS INTO THE DUODENUM

SIR,—I was interested in Mr. Pendower's report (June 6) of a case of aneurysm of the abdominal aorta eroding the posterior wall of the duodenum with fatal gastrointestinal hæmorrhage. A further 3 cases are known to me; 2 have been described elsewhere,[2] both cases being admitted for investigation of severe and repeated hæmatemesis and finally succumbing to a massive hæmatemesis.

The third patient, a man of 71, was admitted to hospital for investigation of intermittent attacks of severe right-sided abdominal pain over a period of three weeks, the character and site of the pain being suggestive but not typical of renal colic. Two years previously he had had frequent attacks of angina pectoris culminating in a more protracted attack of chest pain diagnosed clinically as a myocardial infarction. Clinical examination was initially quite unhelpful, and special investigations on the assumption that the pain was of renal origin were carried out, all with negative findings. Barium enema also showed no abnormality. About three weeks after his admission to hospital an ill-defined pulsatile mass became palpable in the central abdomen. The patient collapsed and died suddenly the following night.

Postmortem examination showed massive gastrointestinal hæmorrhage from erosion of a false aneurysm of the abdominal aorta through the posterior wall of the third part of the duodenum. The "aneurysm" was the result of penetration of an atheromatous ulcer through the aortic wall. There was no abnormality of the urinary tract or other significant finding.

While at the moment these cases represent merely interesting but rare causes of gastrointestinal hæmorrhage, continued advances in the field of cardiovascular surgery may in the future make the consideration of a diagnosis of aortic aneurysm of practical importance in the investigation of a patient with unusual abdominal symptoms or painless hæmatemesis.

Pathology Department, Western Infirmary, Glasgow. A. A. M. GIBSON.

1. *Lancet*, 1958, ii, 677.
2. Gibson Graham, J., Milne, J. A. *Glasg. med. J.* 1952, **33**, 320.

HYPOCŒRULOPLASMINÆMIA AND WILSON'S DISEASE

SIR,—Cœruloplasmin (paraphenylenediamine-oxidase, serum-copper-oxidase) activity is absent or very much below normal in serum from patients with hepatolenticular degeneration (Wilson's disease).[1] According to Scheinberg [2] "it would appear that in Wilson's disease the deficiency or absence of cœruloplasmin in the serum is due to a specific inherited inability to synthesize this protein at a normal rate". Hypocœruloplasminæmia is normal as a transient phenomenon in the newborn and is usual in the nephrotic syndrome from loss of the protein in the urine.[3]

Markowitz et al.[4] have referred to an apparently normal individual who had hypocupræmia and hypocœruloplasminæmia over a period of four years and in whom the only detectable abnormalities were a palpable liver and a persistent, unexplained, neutrophilic leucocytosis. More recently Lygren et al. [5] reported a case of Wilson's disease diagnosed biochemically before clinical manifestations appeared; no neurological signs, mental symptoms, or Kayser-Fleischer rings were present, though there was a family history of Wilson's disease.[6] Lygren referred to a patient reported by Enger who had a normal serum-cœruloplasmin level in the presence of hypercupruria, Kayser-Fleischer rings, and necropsy findings confirming the diagnosis of Wilson's disease.

Since April, 1957, the serum-cœruloplasmin levels of 6 patients with Wilson's disease (5 Chinese, of whom 2 were siblings, and 1 Englishman) have been studied by the simple colorimetric method of Ravin,[7] using paraphenylenediamine as substrate. All estimations were done in duplicate and an EEL photoelectric colorimeter was used for measurement of the colour developed after incubation. All determinations where duplicates did not agree to within 0·1 of a scale division were repeated. No standard was used, the colorimeter readings being referred to directly as units of activity. In all 6 patients cœruloplasmin activity was absent or very low (0·1–1·0, average 0·4 units). During the same period, 298 samples from "normal" individuals were also studied. These were obtained from healthy blood-donors (170) attending the blood-transfusion service (through the courtesy of Dr. M. M. H. Gibson-Hill) and from specimens submitted for Wassermann or Kahn tests (128) to the Serology Section, Department of Pathology, General Hospital, Singapore, from inmates of the Woodbridge Mental Hospital, Singapore (through the courtesy of Dr. L. S. da Silva and Mr. A. Sabaratnam).

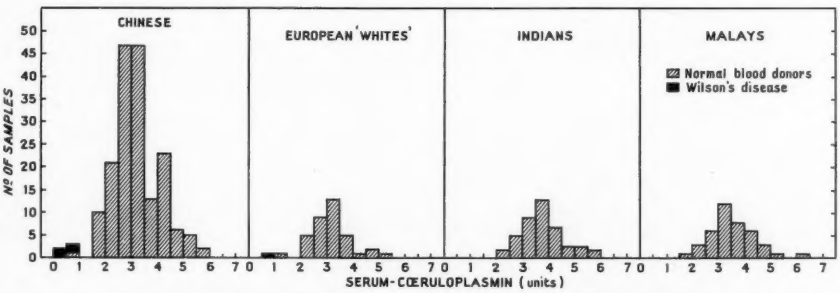
AVERAGE VALUES FOR SERUM-CŒRULOPLASMIN ACTIVITY IN VARIOUS ETHNIC GROUPS

—	European		Indian		Malay		Chinese	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Normal blood-donors	37	3·18	30	3·81	28	3·46	75	2·90
Kahn-test bloods	..	..	14	3·95	13	3·90	101	3·49
Total	37	3·18	44	3·86	41	3·51	176	3·24
Wilson's disease	1	0·65	..	..	..	..	5	0·40

(a) number of samples studied.
(b) average of determinations, in "units".

The accompanying figure shows the frequency distribution of the results by ethnic group. Though the averages (see table) for the Kahn-test samples were slightly higher than those for the blood-donor samples in any one ethnic group, the frequency distributions of both were essentially similar.

Amongst the blood-donors, two had values (1·35 and 0·95 units) which were much below those expected for normal individuals. The first was an adult Englishman of 23 serving with H.M. Forces in Singapore; he was enjoying good general health and was symptom-free. The second was a Chinese male of 18 who was clinically normal (liver not palpable) and nothing in his family history suggested the presence of hepatic disorder or Wilson's disease (Dr. E. Hanam examined this young man clinically). It was not possible to carry out family studies in either of these cases.



Frequency distribution of serum-cœruloplasmin activity by ethnic group.

These results together with the findings of Markowitz et al.,[4] Lygren et al.,[5] and Enger, raise two important questions: is hypocœruloplasminæmia after the first few days from birth always indicative of Wilson's disease, incipient or manifest?; and does a normal serum-cœruloplasmin level ever exclude this diagnosis? The possibility has to be considered that hypocœruloplasminæmia may be present apart from Wilson's disease. Lygren's comment that the diagnostic criteria of hepatolenticular degeneration may have to be reviewed is timely.

The technical assistance of Mr. Stephen Pang in carrying out the cœruloplasmin determinations is gratefully acknowledged.

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F. VELLA.

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2. Scheinberg, I. H. Quoted by Bodansky, O. *Ann. N.Y. Acad. Sci.* 1958, **75**, 380.
3. Vella, F. *Med. J. Malaya*, 1957, **75**, 380.
4. Markowitz, H., Gubler, C. J., Mahoney, J. P., Cartwright, G. E., Wintrobe, M. M. *J. clin. Invest.* 1955, **34**, 1498.
5. Lygren, T., Sorensen, E. W., Bernhardsen, A. *Lancet*, Feb. 7, 1959, p. 276.
6. Lygren, T. *ibid.* p. 275.
7. Ravin, H. A. *ibid.* 1956, i, 726.

In England Now

A Running Commentary by Peripatetic Correspondents

In the Garden.—X

FOR STUDENTS ONLY

Lady Macbeth was a murderess
And not an F.R.H.S.
Had she been the latter
All that natter
About perfumes of Araby and out damned spot
Would not
Have shown her up as a bloody-minded schemer
But just that she was worried about her roses and *Actinonema.*

Obituary

JAMES L. GAMBLE

M.D. Harvard

Dr. James L. Gamble, emeritus professor of pædiatrics at Harvard University, died last month in Boston, Mass. He was known throughout the world for having introduced scientific measurement in pædiatrics and for having established the importance, in relation to treatment, of changes in the extracellular fluid.

A friend writes:

"When Robert Loeb presented the Kober medal of the Association of American Physicians to Dr. Gamble in 1951 he said that 'Beginning with Allan Butler, his first protégé in Boston, there has passed through Dr. Gamble's laboratory a steady stream of embryonic investigators, matured by an indoctrination into scientific critique and enriched by their association with Jim'.

"The most striking point about Dr. Gamble was that he always tried to be sure that anything he did would do no harm, whether to child or adult. The delay in the application to clinical medicine of the observations which he made with Butler and McKhann on the potassium loss in the stools of infants with diarrhœa was due to a great humility; for he knew that the death roll from gastroenteritis had been very considerably reduced by the wise use of solutions of glucose and sodium chloride, and he was fearful lest the giving of intravenous potassium would lead to the death of an infant who might otherwise have recovered.

"An important piece of equipment in the old lab. in Boston was a couch on which he used to rest and think during the afternoon. I remember a not-so-young American commenting that no other place in the United States could survive doing so little work and producing so few papers—by the end of his time there this eager beaver had learnt how quality was more important than quantity and that it is better to make only an occasional observation and have time to think about it than to make thousands of comparatively aimless ones. Much of the interchange of ideas took place at the Thursday tea-parties round the table in the library; at these informal meetings even the most stupid felt at ease and was able quietly to learn.

"Although during the last fifteen years the output from his own pen appeared small, the manuscripts which he rewrote for others in his firm, clear, rather laborious writing were almost countless. They approached in number those men at Harvard to whom he gave unseen help—usually the recipient of a grant had no idea of the source.

"Dr. Gamble took time to get to know a person, but once he had formed a friendship it was for years; he often talked of his former associates—most frequently of Kerpel-Fronius, who had been his intellectual partner in the early 1930s.

"He hated public speaking; and when he was invited to receive the Moxon Award of the Royal College of Physicians, he first wrote to make certain that he would not have to give a speech; on this condition, he was delighted to visit London.

"He was at his happiest in his little log cabin in Maine or sailing one of his S-boats off the coast—then his agility and sureness of movement were quite remarkable, because for years he had looked so frail and hesitant in the rush of an American city. He liked his island, his farm, his pedigree cows, but most of all giving young people the opportunity to enjoy themselves."

CORRIGENDUM: *The Effectiveness in Man of Human Growth Hormone*.—In this article (Jan. 3, p. 7) there was an error in the scale of fig. 1a. The ordinate representing urinary calcium excretion read from above downwards: +0·6, +0·4, and +0·2. In fact this scale should have read: +0·06, +0·04, and +0·02.

News

Royal College of Surgeons of England

At a meeting of the council on June 10, with Sir James Paterson Ross, the president, in the chair, Dr. Paul R. Hawley, Director of the American College of Surgeons, was admitted to the honorary fellowship. Dr. A. H. Bennett, Dr. S. F. L. Dahne, Sir Joseph Pierre, and Prof. A. J. E. Cave were admitted to the fellowship.

Diplomas of fellowship were granted to the following:

*J. D. Thompson, A. J. Woolf, H. S. Brown, R. S. Rihan, †Mary J. Starbuck, D. B. Clarke, L. N. Allen, I. C. Cree, J. W. Maltby, V. Anomah Ngu, K. P. Robinson, D. W. Daly, M. R. H. Ashken, J. D. Atwell, D. A. Packham, Idris H. Foy, P. W. Van Gelderen, R. N. Austin, *D.W. Stuart, †C. W. Thornhill, †Satya Bhushan Kapur, A. W. Bruce, *Turabally Fazleabas, Ranjitsingha Lakshman Thambugala, G. A. Court, I. H. Leitch, Mini Hoshang Mehta, Bhalchandra Vasudeo Nabar, P. M. H. Rack, E. T. Heath, A. W. Jansz, P. M. Jayawardena, D. S. Leitch, Jack Magell, A. C. Townsend, W. A. Crabbe, B. K. Hope, C. O. Ovendale, Louis Solomon, Lindsay Symon, T. G. Thomas, Puspanathan Sadasivan Vaithilingam, W. M. Wearne, John Anderson, V. Ariyaratnam Benjamin, R. R. Cooke, R. C. Hartley, E. R. Hitchcock, E. I. Kohorn, Elsa Little, Abbas Ahmedally Lookman, M. R. Madigan, B. B. Mandell, D. G. Millar, Samuel Movsas, D. A. O'Rourke, Susila Rajagopalan, R. A. Sellwood, Vasantlal Gulabchand Shah, Leslie Turner, Purushottam Upadhyaya, W. G. Williams, R. T. Aldridge, Braj Nandan Singh Bhonsla, J. C. Bremner, J. M. Collibee, †B. D. Coote, A. M. Cuthbertson, Dharam Rai, *Dallas Finney, Ajit Kumar Ghosh, Raj Kumar, D. N. Leckie, Ernest Lewis, W. A. A. G. Macbeth, P. R. MacNeil, J. C. D. Mill, Subbarayan Keshava Murthy, G. D. Oates, E. G. Roberts, Joe Siegenberg, G. W. G. Sinclair, *G. D. L. Smyth, C. M. Vickery, Soon Kai Wong, Uday Shankar Arora, Kong Thoe Chan, A. M. Chirnside, B. W. Fox, W. A. Gokavi, R. J. Healey, C. H. Hooker, Po Yat Iu, M. J. Killingback, *W. S. Lund, J. E. Moulton, Vinayak Nagesh Shrikhande, P. K. Steedman, David Stein, Walid Schowkat Al-Khayal, I. J. Cour-Palais, P. D. Hughes, M. X. Shanahan, T. K. F. Taylor, Marx Wald, Kean Seng Yeoh.

* In otolaryngology. † In ophthalmology.

Dr. R. H. P. Fitzpatrick and Dr. G. A. Haydock were admitted to the fellowship in the Faculty of Anæsthetists.

Royal College of Physicians of Ireland

At a meeting of the college on June 5, with Dr. P. T. O'Farrell, the president, in the chair, the following were admitted to the fellowship:

J. B. Lyons, D. E. Mellon, O. C. Ward, M. K. O'Driscoll, E. E. Doyle, M. J. M. Solomons, James Hingston (*in absentia*).

The following were admitted as licentiates and members:

M. T. N. Callaghan, James Hunter, D. N. Golding, Edward Tempany, I. J. Dalrymple, E. E. Clarke.

Ciba Foundation

The Ciba Foundation lecture on research on ageing is to be delivered this year by Dr. A. C. Upton, of Oak Ridge National Laboratory, Tennessee, who will speak on Radiation and Ageing. The lecture is to be given at St. Bartholomew's Hospital Medical College, London, E.C.1, at 2.30 P.M. on Thursday, July 2. Tickets may be had from the assistant secretary of the Foundation, 41, Portland Place, London, W.1. An announcement of this lecture appeared incorrectly in the issue of June 6.

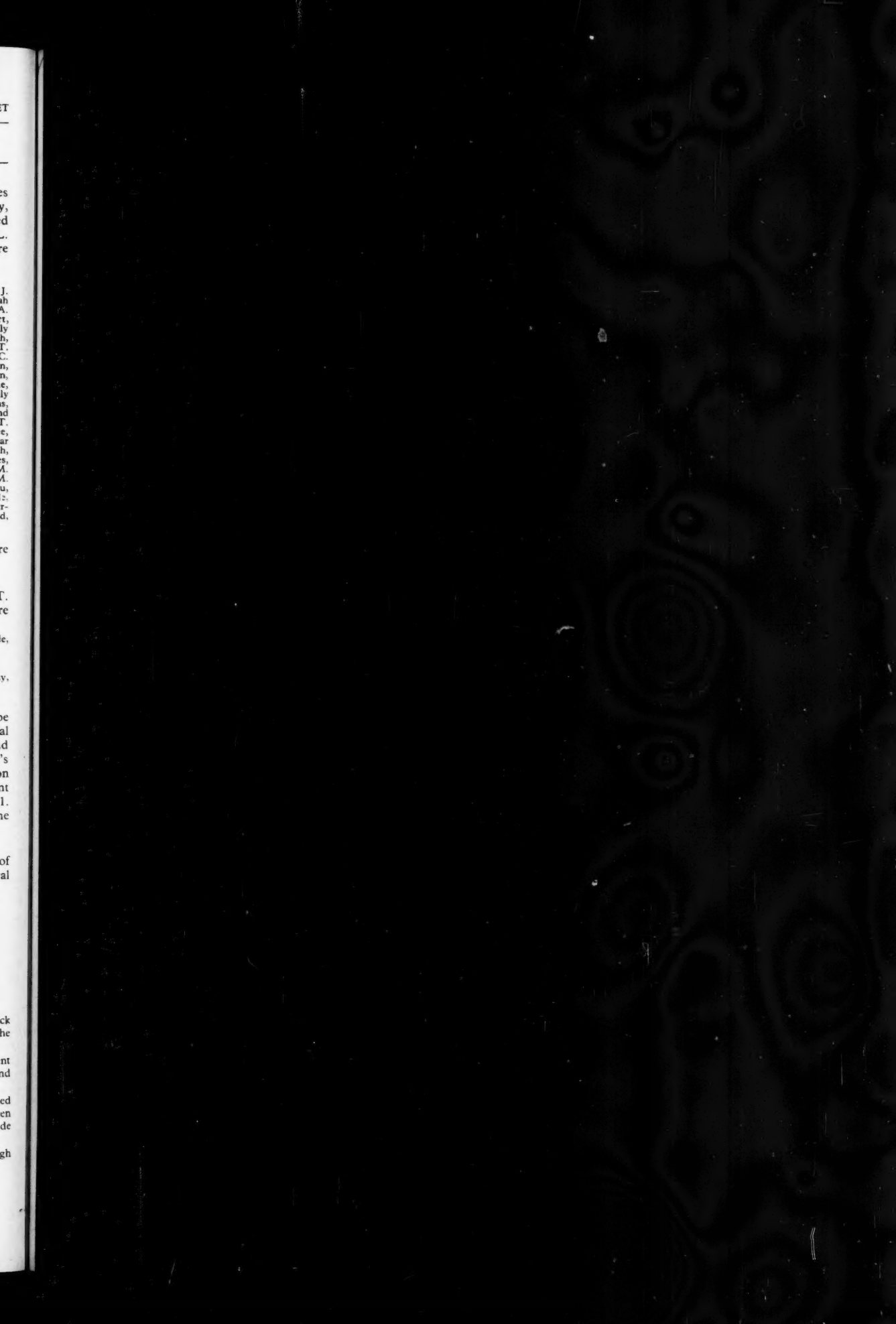
Merit Awards

At the end of 1958 in England and Wales the distribution of merit awards between consultants in general medicine, general surgery, and mental health was as follows:

Specialty	Number of consultants	Awards			
		A	B	C	Total
General medicine	838	61	155	259	475
General surgery ..	862	59	144	256	459
Mental health ..	631	7	23	63	93

Mr. H. F. Rutherford, house governor of The Hospital for Sick Children, Great Ormond Street, has retired after 43 years in the hospital service.

The East Anglian Thoracic Society has resolved that urgent consideration be given to admitting to this country, treating, and resettling tuberculous refugees and their families.

The papers given at the C.I.O.M.S. conference on controlled clinical trials (see *Lancet*, April 4, 1959, p. 739) have now been mimeographed. Copies may be had from the C.I.O.M.S., 2, place de Fontenoy, Paris, 7ème, France.

The address of the Muscular Dystrophy Group is now 26, Borough High Street, London, S.E.1.